	Day (n = 120)	Night (n = 198)	p
Age (years)	59 ± 13	60 ± 14	NS
Males	79%	79%	NS
Anterior MI	49%	51%	NS
Diabetes mellitus	11%	12%	NS
Multivessel disease	39%	42%	NS
Time to admission (min)	173 ± 83	185 ± 88	NS
PTCA Success*	93%	94%	NS
Time to reperfusion (min)	220 ± 85	241 ± 92	NS
Emergency CABG (%)	0	0.6%	NS
Ventricular tachyarrythmia	8.0%	5.1%	NS
Left ventricular EF (%)	53 ± 12	51 ± 13	NS
TIMI 3 at discharge	96%	98%	NS
In-hospital death	5.3%	6.9%	NS

*TIMI 3 flow and <50% residual stenosis

Conclusion: In thrombolysis-eligible Pts, baseline characteristics and outcome of primary PTCA appear similar at night and during the day. There is no reason to treat Pts differently during the "off" hours.

926 Left Ventricular Remodeling After Infarction

Monday, March 17, 1997, Noon–2:00 p.m.
Anaheim Convention Center, Hall E
Presentation Hour: 1:00 p.m.–2:00 p.m.

MONDAY POSTER

926-25 Early Revascularization of Asymptomatic Patients With a Totally Occluded Infarct-Related Artery Improves Left Ventricular Remodeling

G. Dangas, M.E. Goldman, A. Cohen, V. Kakarala, S.K. Sharma, S. Duvvuri, T.P. Cocke, J.D. Marmur, J.A. Ambrose. *The Cardiovascular Institute, Mount Sinai Medical Center, New York, NY, USA*

While symptomatic patients post Q-wave MI benefit from early elective revascularization, it is unknown whether asymptomatic patients improve, including those with a totally occluded infarct-related artery. We prospectively studied 20 consecutive asymptomatic patients (age 55 ± 2 years, 18 males) following a Q-wave MI (CK 3235 ± 450, 16 anterior) with ≥70% stenosis of the infarct-related artery who underwent elective revascularization (day 3–7 post-MI). Baseline rest echo was performed ≤ 24 hrs before revascularization, and repeat echo at 8-week follow-up. All studies were analyzed blindly by two-reader consensus, and left ventricular volumes and ejection fractions (EF) were measured by Simpson's rule (from apical 4-chamber view).

Results: Compared to patients with patent infarct-related arteries, those with totally occluded arteries had greater end-systolic volume (117 ± 47 ml vs. 68 ± 8 ml) and lower EF (39 ± 6% vs. 44 ± 2%) at baseline (p = ns). Patients with total occlusions (TIMI flow 0 or 1) had significant changes in end-systolic volume and EF (mean ± SE) after revascularization compared to patients with patent arteries (TIMI flow 2 or 3):

ΔPost – Pre	TIMI 0–1 (n = 5)	TIMI 2–3 (n = 15)	p
ΔSystolic volume	−22.2 ± 17.1ml	5.1 ± 3.4ml	0.02
ΔEF	13.6 ± 7.1%	0.7 ± 2.2%	0.03

Conclusion: Global left ventricular remodeling, as assessed echocardiographically, is decreased after elective revascularization early post Q-wave MI in asymptomatic patients with totally occluded infarct-related arteries. Larger randomized studies should further investigate this important observation.

926-26 Coronary Flow Reserve in Infarct Related Artery and Left Ventricular Remodeling in Patients with Anterior Myocardial Infarction

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The previous reports have demonstrated that preserved coronary flow reserve (CFR) in the infarct region is associated with the improvement of wall motion abnormality after reperfusion in acute myocardial infarction. To investigate the relationship between residual CFR in the infarct related artery and subsequent left ventricular (LV) remodeling and function, we studied 36 patients (pts, mean age: 55 ± 11 years, 29 males, 7 females) with the first anterior myocardial infarction. Twenty pts received thrombolytic therapy. Coronary angioplasty was performed in 25 pts 7–10 days after infarction and mean residual stenosis was 37 ± 25%. CFR was measured distal to the infarct lesion by intracoronary Doppler wire (0.014-inch) and injection of adenosine after angioplasty. Pts were divided into two groups with CFR < 1.3 (n = 13) or ≥1.3 (n = 23). End diastolic (EDV) and end systolic (ESV) LV volumes and ejection fraction (EF) were measured by two dimensional echocardiography and radionuclide angiography 7–10 days (after angioplasty) and 3 months post infarction.

At 3 month follow-up	CFR <1.3 (n = 13)	CFR ≥ 1.3 (n = 23)
% ΔEDV	24.8 ± 7.8	0.1 ± 4.2*
% ΔESV	14.1 ± 6.8	−11.1 ± 12.5*
ΔEF	−2.3 ± 4.7	6.4 ± 6.3

(*p < 0.05)

Thus, patients with poor CFR (<1.3) as directly measured in the infarct related artery have more LV dilation than those with relatively preserved CFR. Therefore, CFR may be a reflection of coronary microvascular integrity which may have a significant impact upon LV remodeling process after myocardial infarction.

926-27 Small Myocardial Infarction may Cause Late Ventricular Dilation and Dysfunction Possibly due to Chronic Infarct Expansion

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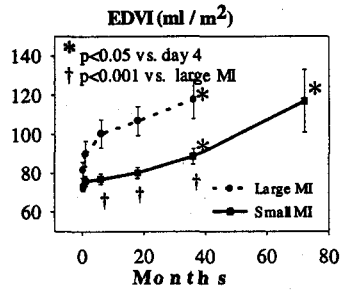
Left ventricular (LV) dilation after myocardial infarction (MI) is an independent predictor of adverse events and mortality. Asymptomatic patients with small MI are considered to be at low risk for LV dilation and late dysfunction. We prospectively followed 104 asymptomatic normotensive patients after first MI without ischemia or reinfarction for 7 years. At baseline, MI size (cineangiography) was 7 ± 1% (mean ± sem) and 30 ± 2% and radionuclide ejection fraction (EF) 53 ± 2% and 30 ± 2% in small (n = 78) and large (n = 27) MI, respectively. From 4 days (D), 4 weeks (W), 0.5 years (Y), 1.5 Y, 3 Y until 7 Y after MI, LV end diastolic volume index (EDVI), by gated SPECT increased. This slow LV dilation (figure) in small MI was followed by late deterioration of EF (4 D: 53 ± 2; 4 W: 54 ± 2; 1.5 Y: 53 ± 2; 3 Y: 49 ± 2; 7 Y: 34 ± 3†; † ANOVA p < 0.05 vs. 4 D). Repeat angiography 72 months after MI (n = 9) revealed an increase in end-diastolic circumference (4 W: 27.6 ± 0.6; 72 M: 31.0 ± 0.7 cm; p = 0.0119) due to increased MI segment length (4 W: 1.3 ± 0.4; 72 M 7.3 ± 1.1 cm; p = 0.0005) at unchanged noninfarcted segment length (4 W: 26.1 ± 0.6; 72 M: 24.7 ± 1.8 cm; p = 0.4606).



Thus, even after small MI, the LV may undergo slow but long-term remodeling which appears to be due to chronic infarct expansion.

926-28 Progression of Left Ventricular Dilation Following First Q-Wave and Non Q-Wave Myocardial Infarction

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In this prospective study, we examined the extent of LV chamber dilation, a measure of LV remodeling, following first Q-wave and non Q-wave (NQ) acute myocardial infarction. A group of 165 patients with infarction, 120 with Q and 45 with NQ based on pre-hospital discharge ECG were stud-

	Baseline	3 M	6 M
Q-EDVI (ml/m²)	56 ± 16	64 ± 16*	68 ± 16†
NQ-EDVI (ml/m²)	55 ± 15	63 ± 16*	61 ± 15†‡
Q-ESVI (ml/m²)	27 ± 10	32 ± 12*	33 ± 11†
NQ-ESVI (ml/m²)	25 ± 10	28 ± 8*	28 ± 10†‡

*†‡P < 0.001. * Baseline vs. 3 M; †Baseline vs. 6 M; ‡Q vs. NQ]

ied. Two-dimensional echocardiograms were obtained within 48 hours of hospital admission (baseline), and at 3 and 6 months (M) after hospital discharge. Echocardiograms were used to calculate LV end-diastolic volume index (EDVI) and end-systolic volume index (ESVI).

There were no differences in baseline EDVI and ESVI between Q and NQ infarction patients. EDVI and ESVI increased after 3 M and 6 M of follow-up in both Q and NQ patients. At 6 M, both EDVI and ESVI were significantly greater in Q compared to NQ patients. *Conclusions:* Progressive LV chamber enlargement occurs following both Q and NQ acute myocardial infarction. The extent of LV enlargement, however, is greater in Q than in NQ infarction possibly as a result of greater loss of viable myocardium.

926-29 Is Anterior Wall Involvement in Non-Q Wave Myocardial Infarction Associated With Left Ventricular Dysfunction?

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Previous studies have shown that anterior Q-wave MI patients have worse LV function and poor outcome. We postulated that non-Q wave MI patients with anterior wall involvement would have poorer LV function than those with ischemia in other areas. Accordingly, we prospectively evaluated the relationship between perfusion (Tl^{201} scintigraphy) and function abnormalities (multigated radionuclide ventrigraphy, RVG) performed on days 5–7 after a non-Q wave MI in the Veterans Administration Non-Q Wave Infarction Strategies in Hospital (VANQWISH) study. Of the 389 pts evaluated, hypoperfusion in various segments was noted as follows: anterior, 58 pts (15%); septal, 55 (14%); posterolateral, 55 (14%); inferior, 114 (29%); posterior, 107 (27%), and lateral, 134 (34%). For the whole group, the mean LVEF was 49.6 ± 14%. The correlation between impaired (EF < 50%) or preserved (EF ≥ 50%) LV function revealed a significant correlation between anterior ($\chi^2 = 6$, $p = 0.01$) and septal ($\chi^2 = 5.6$, $p = 0.02$) involvement but not for other areas. The evaluation of regional wall motion abnormalities (WMA) also showed a strong correlation between anterior hypoperfusion and presence of anterior WMA ($\chi^2 = 15$, $p = 0.000$) and septal hypoperfusion and septal WMA ($\chi^2 = 8$, $p < 0.005$) but no significant relationship was found for other areas.

Conclusion: These findings indicate that, despite lack of Q waves, anterior and septal perfusion abnormalities in patients with non-Q wave MI are correlated with impaired LV function, as indicated by lower LVEF and the associated regional wall motion abnormalities.

926-30 C-Reactive Protein as a Predictor of Infarct Expansion and Cardiac Rupture After a First Q Wave Acute Myocardial Infarction

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Pump failure following acute myocardial infarction (AMI) can be predicted by several indices which estimate infarct size. However, there are few indices that predict infarct expansion and cardiac rupture. We focused on the prognostic significance of serum C-reactive protein (CRP) after AMI. Serum CRP levels were measured every 24 hours in 220 patients with a first Q-wave AMI. In-hospital complications, predischarge left ventriculographic findings, and long-term prognosis were assessed in relation to peak CRP levels. Lower peak CRP levels were associated with the presence of preinfarction angina, with successful revascularization, and with age <70 years. Peak levels of both CRP and creatine kinase (CK) were higher in patients with pump failure than in those without pump failure. In patients with cardiac rupture, peak CRP levels were higher than in those without rupture (23.7 ± 3.9 vs 12.2 ± 10.6 mg/dl, $p = 0.001$); peak CK levels were not predictive. Peak CRP levels correlated positively with left ventricular (LV) end-diastolic volume ($p = 0.002$) and inversely with ejection fraction ($p = 0.0001$). Higher CRP levels were found in patients with LV aneurysm ($p = 0.001$ vs those without), aggravated heart failure during the first one year after AMI ($p = 0.03$ vs those without) and one-year cardiac death ($p < 0.0001$ vs survivors). Multivariate analysis confirmed that an elevation of the peak CRP level above 20 mg/dl was an independent predictor of cardiac rupture (relative risk, rr = 4.72, $p = 0.004$), left ventricular aneurysm formation (rr = 2.11, $p = 0.03$), and one-year cardiac death (rr = 3.45, $p = 0.0001$). *Conclusions:* Cardiac rupture, left ventricular aneurysm formation and one-year cardiac death were associated with an elevation of serum CRP early after AMI, suggesting that elevation of CRP levels after AMI may predict infarct expansion.

927 Stents: Subacute Thrombosis

Monday, March 17, 1997, Noon–2:00 p.m.
Anaheim Convention Center, Hall E
Presentation Hour: Noon–1:00 p.m.

927-1 Use of Cilostazol, a Novel Antiplatelet Agent, as a Post-Palmatz-Schatz Stenting Regimen

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Background: Ticlopidine achieved widespread use as an antiplatelet agent following Palmatz-Schatz stent (PSS) implantation. However, this drug is sometimes associated with serious side effects (including leukocytopenia and liver dysfunction). Cilostazol, a potent inhibitor of phosphodiesterase, is a novel antiplatelet agent which is currently available in Japan and being tested in clinical trial in the United States for peripheral artery disease. Previous studies have indicated that, as compared with ticlopidine, cilostazol achieves similar antiplatelet effects with less frequent complication rates. In this study, we used cilostazol as an antiplatelet agent following PSS implantation to investigate the feasibility of preventing stent thrombosis. *Methods:* A total of 60 patients (pts; 48 men, mean age 60 ± 9) with angiographic success following PSS was involved. Seven pts received multivessel stenting, and 21 received overlapping stenting. Sixty-seven lesions (including 36 LAD, 7 LCx, 23 RCA, and 1 SVG) were treated with PSS. All lesions were expanded by using high-pressure balloons with a final balloon pressure of 18.6 ± 1.8 atm under the guidance of quantitative coronary angiography. Following PSS, these pts received cilostazol 200 mg/day along with aspirin 243 mg/day. Heparin was discontinued immediately after stent implantation. Coumadin was not given either prior to or post-implantation. *Results:* One month after stent implantation, follow-up coronary angiography was performed in all 60 pts (100%). During this period, none developed stent thrombosis, acute myocardial infarction, CABG, or serious side effects (including leukocytopenia and/or liver dysfunction). The minimum lumen diameter was 0.86 ± 0.48 mm before PSS, 2.91 ± 0.36 mm immediately after PSS, and 2.65 ± 0.39 mm at 1 month after PSS. *Conclusions:* These results suggest that cilostazol may be potentially useful as a new antiplatelet agent following PSS implantation and may have a similar preventive effect on stent thrombosis and less side effects than does ticlopidine.

927-2 The MUST Trial. In-hospital and Clinical Events at Six Months. Final Results

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The MUST trial was designed to validate the concept developed by the French Registry of coronary stenting with the association of Ticlopidine and Aspirin as sole post stenting treatment.

Population: from January to August 1995, 260 pts were included in an open, prospective, multicenter, observational study. Patients selected had stable or unstable angina, a single lesion in a native artery treated by elective stenting with a single 15 mm Palmaz Schatz stent. Medical therapy: the patients received 100 mg Aspirin and 250 to 500 mg of Ticlopidin daily for one month after the procedure.

Procedure: 3 (1.15%) stenting procedures failed (2 cross over to a Wiktor stent), 3 (1.15%) stents were lost.

In-hospital events: no patient died, 1 (0.4%) underwent emergency surgery, 6 (2.1%) had an MI, 3 (1.15%) showed subacute thrombosis and 5 (1.9%) underwent repeat PTCA during the hospital phase.

Bleeding complications included 1 (0.4%) gastrointestinal bleeding and 1 (0.4%) groin hematoma requiring blood transfusion. Mean in-hospital stay was 3.4 days.

At 6-month follow-up: There were no late death or MI and 4 patients (1.5%) underwent CABG. Fifteen (5.8%) had repeat PTCA at the same site.

In conclusion: coronary stenting with low doses of 2 antiaggregants seems to be feasible and efficient. The in-hospital event rate is acceptable and the 6 month event rate seems comparable with those of the stented arm of the Benestent studies.